

Usefulness and limitations of circumferential fibre shortening velocity in evaluating segmental disorders of left ventricular contraction

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In order to study the possibility of using circumferential fibre shortening velocity (V_{CF}) to assess regional left ventricular performance, 21 left ventricular angiograms (single-plane cine) from 19 patients were examined. In 8 patients with normal left ventricle, measurements of instantaneous and mean V_{CF} in 4 equidistant chords revealed uniform velocities at all chords. In 7 patients with coronary artery disease mean and max V_{CF} were depressed in the areas involved by asynergy; however, when only one wall was affected, velocity measurements of the hemichords provided a better definition of regional performance. V_{CF} at the base (chords A and B) was normal in 2 out of 3 patients with apical asynergy, and augmented in one. Angiotensin produced a reduction in the mean velocity of the anterior wall in a patient with inferior dyskinesis-akinesis; in contrast, nitroglycerin was associated with augmented velocity of the non-akinetic segment in a second patient with inferior akinesis and anteroapical hypokinesis.

In contrast to localized asynergy, a diffuse reduction in V_{CF} was observed in a patient with cardiomyopathy. V_{CF} at the base was reduced in a patient with mitral stenosis and in another with mitral valve prolapse, indicating the presence of a localized basal abnormality of left ventricular contraction. The time course of V_{CF} in the patient with mitral prolapse showed an early peak before aortic valve opening, corresponding to a reduction in left ventricular volume (ΔV) of 22 per cent (in contrast to 3 per cent reduction in the normal left ventricle). Fibre shortening before aortic valve opening was also present in chronic mitral regurgitation ($\Delta V = 15\%$).

Segmental analysis of normalized velocities provides a quantitative approach to the evaluation of regional left ventricular performance. The sensitivity of V_{CF} to acute changes in loading, however, may limit in some instances the usefulness of this index.

The search for a sensitive and simple method to measure the inotropic state of left ventricular myocardium has been a continuing and important one. Determination of instantaneous tension-velocity relations throughout ejection provides a sensitive means of comparing the level of the ventricular contractile state among patients (Gault, Ross, and Braunwald, 1968). However, such analysis requires frame by frame measurements of left ventricular dimensions, accurate estimations of

changes in ventricular wall thickness, and high fidelity left ventricular pressure recordings. In contrast, it has been shown that the mean velocity of circumferential fibre shortening (V_{CF}) provides a relatively simple and satisfactory measurement of left ventricular performance, even when valvular and myocardial defects coexist (Karliner *et al.*, 1971b). Quantitative comparison of the left ventricular performance among patients is also possible since velocity is divided by end-diastolic circumference and expressed per unit of circumferential length. The applicability of this method has been based on the assumption that mean V_{CF} at the minor equator reflects the inotropic state of the entire ventricle, an assumption precluded by the presence of segmental myocardial disease. The purpose of the present study is to examine the usefulness and

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limitations of segmental measurements of V_{CF} in the evaluation of localized disorders of left ventricular contractility.

Methods

Left ventricular cineangiograms obtained from 19 patients, 21 to 59 years of age, were analysed. One group (8 patients) had normal haemodynamics at rest, normal synergy of left ventricular contraction, and normal coronary arteriograms. A second group (11 patients) consisted of 7 cases of coronary artery disease with abnormal left ventricular performance and regional disorders of wall motion, 3 patients with valvular lesions (mitral stenosis, mitral regurgitation, and mitral prolapse), and one patient with congestive cardiomyopathy. The patients included in this report were selected on the basis of representing a portion of a disease spectrum; they were not necessarily considered representative or average cases. High quality left ventricular cineangiograms were required (adequate left ventricular opacification without premature contractions). All patients were in normal sinus rhythm.

Diagnostic right and left heart catheterization was performed in the post-absorptive state. Left ventricular angiograms (single-plane cine, right anterior oblique) were obtained, with injection of 45 to 55 ml contrast material (Renografin 76) through a multiple side-holes catheter at a film speed of 60 frames/second. A second left ventricular angiogram was performed in 2 patients: in 1 of them the angiogram was done 2 minutes after the administration of nitroglycerin (0.8 mg sublingual); in the other patient, the left ventricular angiogram was performed during an infusion of angiotensin (2 µg/ml in 5% D/W) at a rate sufficient to produce a distinct pressor response. The ventriculograms were inspected frame by frame; end-diastole was defined as the earliest frame to show maximum endocardial outward excursion (maximum volume). Aortic valve opening, identified by observation of the valve leaflets motion, occurred 4 ± 1 frames (67 ± 17 msec) after end-diastole; therefore, the fifth frame following end-diastole was selected as the onset of left ventricular ejection in all cases. End-systole was defined as the frame showing maximal endocardial inward motion (minimum volume). The left ventricular ejection time (LVET) was determined by the number of frames from the onset of ejection to end-systole. In the patient with mitral regurgitation, and in the mitral valve prolapse, the period from end-diastole to aortic valve opening was included in the calculation of the ejection time. The left ventricular cavity was drawn at end-diastole, and every fourth frame throughout systole. In each drawing a long axis was constructed from the ventricular apex to the angle formed by the profile of the mitral valve and the posterior sinus of the aortic valve. Five equidistant chords joining the anterior endocardial surface to the inferior surface were drawn perpendicular to the long axis. The chords were identified as A to E from the base to the apex. Analysis of the velocity of chord E was not performed because of the apparent absence of this chord at end-systole (apical cavity obliteration) in several normal ventricles. Chords A to D can be seen in the angiogram shown in Fig. 5.

The mean circumferential fibre shortening rate (mean V_{CF} in circumferences/sec) for each chord was calculated as

$$\text{mean } V_{CF} = (D_d - D_s) / D_d \times \text{LVET}$$

where D_d and D_s represent the chord or diameter at end-diastole and at end-systole, respectively. In 4 patients, the mean shortening velocity of the anterior and posterior hemichords (radii) was also calculated by substituting radius for diameter in the above formula. 'Instantaneous' circumferential fibre shortening velocity was determined every 67 msec (every 4 frames) as

$$V_{CF} = (D_1 - D_2) / D_1 \times 0.067,$$

where D_1 = initial chord length and D_2 = final chord length. Measurements were started at end-diastole and carried on every fourth frame throughout systole. The systolic ejection fraction was determined using the method of Greene *et al.* (1967). In selected cases (mitral regurgitation and mitral prolapse) left ventricular volume was determined at end-diastole, at the time of aortic valve opening, and at end-systole. Dimensions were corrected for magnification and distortion by means of a reference (1 cm squares) grid. Total stroke volume was taken as the difference between end-diastolic and end-systolic volume; regurgitant volume was calculated as the difference between the total stroke volume and the forward stroke volume (determined according to the Fick principle for oxygen).

Results

The data are presented in the Table and in Fig. 1 to 6.

Normal pattern of left ventricular contraction

The normal pattern of contraction was characterized by a uniform inward motion of the ventricular endocardium during systole. As shown in Fig. 1, mean V_{CF} was similar for each chord. Likewise, 'instantaneous' V_{CF} , derived from every fourth cine frame, was similar in each of the four chords (Fig. 2). Peak V_{CF} occurred between 133 and 200 msec after end-diastole. V_{CF} before aortic valve opening averaged 0.2 circ/sec, corresponding to a change in volume of 3 per cent.

Abnormal pattern of left ventricular contraction

Analysis of mean and 'instantaneous' V_{CF} for Cases 9, 10, and 13 is shown in Fig. 1 and 2. A reduction in both mean and peak V_{CF} was found in the chords adjacent to areas showing localized disorders of wall motion. Both mean and peak V_{CF} were well preserved in the non-involved areas of the patients with apical disease (Cases 9, 10, and 11); 2 of these 3 patients had normal ejection fractions. In Case 9 the velocity of fibre shortening in chord B appeared to be greater than that observed in any of the normal hearts (Fig. 1 and 2). As is shown in Fig. 3, mean

TABLE

Case no.	Age/sex	BSA (m ²)	V _{CF} (circ/sec)		Chord A		Chord B		Chord C		Chord D		Clinical diagnoses	SEF
			Max	Mean	Max	Mean	Max	Mean	Max	Mean	Max	Mean		
1	46/M	1.75	3.49	1.59	3.19	1.99	3.49	1.96	4.12	2.04	Left anterior descending artery – right ventricular fistula*		65%	
2	40/F	1.64	2.53	1.18	3.35	1.55	2.45	1.39	3.53	1.58	Chest pain, normal coronary artery		73%	
3	59/M	2.25	3.50	2.42	3.34	2.50	3.76	2.63	2.63	2.12	Chest pain, normal coronary artery		85%	
4	22/M	2.26	1.39	0.78	2.53	1.10	2.61	1.20	2.89	1.04	Functional murmur		60%	
5	26/F	1.83	3.53	1.46	3.55	1.50	3.20	1.46	3.35	1.50	Chest pain, normal coronary artery		82%	
6	21/M	1.84	2.42	1.11	2.33	1.30	2.37	1.22	2.27	1.11	Functional murmur		66%	
7	53/M	2.13			3.19	1.50	2.79	1.47	2.46	1.41	Chest pain, normal coronary artery		60%	
8	43/M	1.67	2.32	1.36	3.53	1.71	3.67	1.73	3.55	1.86	Chest pain, normal coronary artery		64%	
	Mean		2.74	1.41	3.13	1.64	3.04	1.63	3.10	1.58				
	SD		0.54	0.52	0.31	0.42	0.38	0.48	0.43	0.27				
	SEM		0.21	0.20	0.11	0.15	0.20	0.17	0.23	0.14				
9	50/F	1.80	2.49	1.41	5.10	2.06	2.57	1.50	2.10	0.92	Coronary artery disease, apical hypokinesis		67%	
10	59/F	1.60	2.58	1.46	2.46	1.21	0.76	0.51	0.32	0.14	Coronary artery disease, apical akinesis		46%	
11	42/F	1.97	2.45	1.66	2.14	1.41	1.09	1.08	0.88	0.87	Coronary artery disease, apical akinesis		63%	
12	36/M	1.74	1.30	0.91	1.39	0.86	1.15	0.96	2.00	0.84	Coronary artery disease, anterior-apical hypokinesis		47%	
13	49/M	1.88	1.32	0.97	1.19	0.68	1.50	0.39	0.64	0.19	Coronary artery disease, anterior-apical akinesis		34%	
14	48/M	2.05	1.45	0.69	1.57	0.78	2.75	0.87	2.74	1.19	Coronary artery disease, inferior dyskinesis-akinesis		52%	
15	48/M	1.95	0.88	0.67	0.93	0.74	0.87	0.70	0.98	0.62	Coronary artery disease, inferior akinesis + anterior-apical hypokinesis		39%	
16	56/F	1.85	0.40	0.20	0.60	0.21	0.38	0.22	0.18	0.24	Congestive cardiomyopathy		25%	
17	22/M	1.80	1.87	1.02	2.09	0.89	3.24	2.10	3.29	1.90	Mitral stenosis		72%	
18	30/M	2.01	0.80	0.34	1.84	1.60	1.90	1.23	1.78	1.09	Mitral valve prolapse		63%	
19	34/M	1.54	2.22	1.27	2.27	1.34	2.29	1.31	2.82	1.41	Mitral regurgitation		71%	

* Pulmonary to systemic flow ratio < 1:1:1.

Abbreviations: V_{CF}=circumferential fibre shortening rate; BSA=body surface area; Max=maximum; SD=standard deviation; SEM=standard error of the mean. SEF=systolic ejection fraction.

V_{CF} in all 4 chords was reduced in Case 14 (inferior dyskinesis-akinesis) while the mean normalized velocities of the anterior hemichords (radii) were normal. Likewise in Case 12 (anteroapical hypokinesis), mean V_{CF} was reduced in all chords while inferior wall motion was preserved. There was good agreement between circumferential and radial velocities in the normal (Fig. 3). The patient with anteroapical akinesis (Case 13) had generalized reduction in the extent (SEF=34%) and velocities of wall motion (Fig. 1 and 2). This patient qualitatively resembles the patient with congestive cardiomyopathy (Case 16).

In the patient with mitral stenosis (Case 17), both mean and 'instantaneous' V_{CF} were reduced at the base (chords A and B) and normal towards the apex (chords C and D). The systolic ejection fraction was normal (72%) (see Fig. 1 and 4).

A striking reduction in mean and 'instantaneous' V_{CF} was observed at chord A in the patient with mitral valve prolapse (Case 18). Though mean V_{CF} was preserved in the remaining chords, peak V_{CF} was slightly reduced (Fig. 1 and 4). Prolapse of the posterior leaflet of the mitral valve began immediately after end-diastole and reached maximum shortly after aortic valve opening (Fig. 5); prolapse

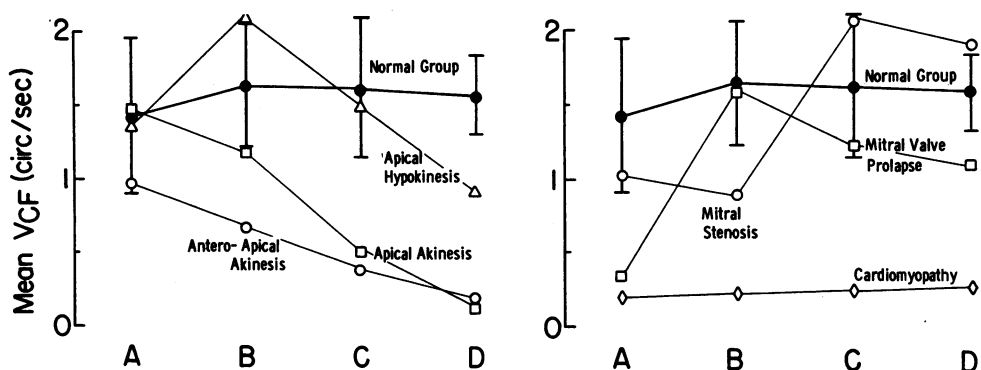


FIG. 1 Mean V_{CF} (average \pm SD) for chords A to D in the normal group and in several patients with asynergy are shown. In the normal left ventricle, V_{CF} is uniform in each of the four chords. Segmental abnormalities of wall motion are associated with reduced V_{CF} in the involved areas.

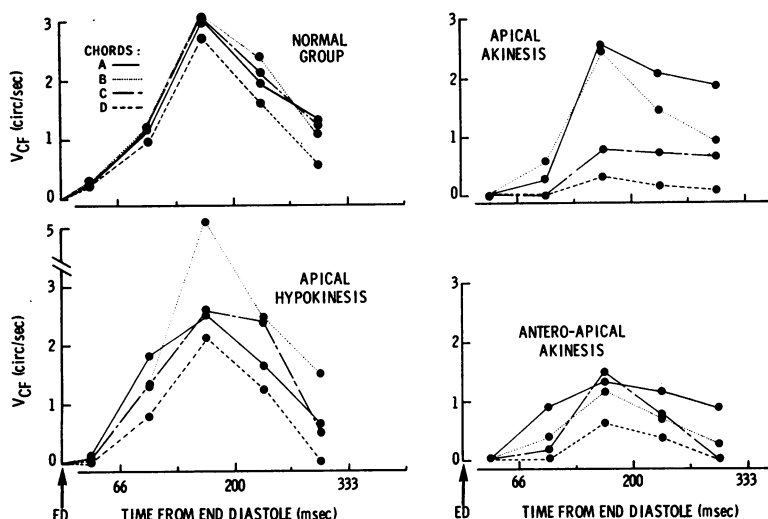


FIG. 2 The 'instantaneous' course of circumferential fibre shortening velocity for the normal group is shown in the panel on the upper left; three individual examples of coronary artery disease with asynergy are also shown. In the normal left ventricle the time course of V_{CF} is similar in each chord. Segmental reductions in V_{CF} are seen in the patients with asynergy. Note the high value for peak V_{CF} at the base (chord B) in the patient with apical hypokinesis.

was accompanied by significant fibre shortening (average $V_{CF} = 1.8$ circ/sec) in chords B, C, and D during the 'isovolumic' period (Fig. 4). In this patient the end-diastolic volume was 90 ml/m², the volume remaining within the left ventricular cavity at the time of aortic valve opening was 70 ml/m², total angiographic stroke volume was 56 ml/m² and the total 'regurgitant' volume was 25 ml/m². No

regurgitation of dye into the left atrium was seen; the 'regurgitant volume' was, therefore, contained within the mitral valve apparatus.

Chronic rheumatic mitral regurgitation, likewise, was associated with volume reduction and significant fibre shortening before aortic valve opening. In this patient regurgitation of contrast into the left atrium began immediately after end-diastole and

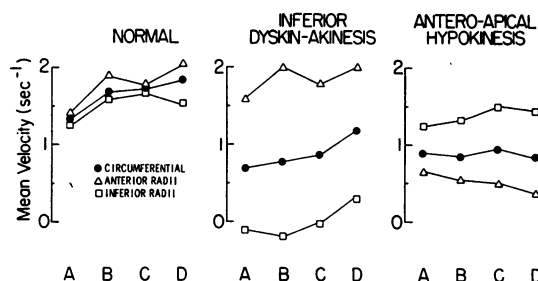


FIG. 3 Mean circumferential fibre shortening velocity and the mean normalized velocities of the hemichords (radii) from a normal (panel on the left) and from two patients with asynergy are shown. In left ventricular asynergy, analysis of the hemichords provides better definition of regional performance than V_{CF} . See text for details.

appeared to continue throughout systole. The end-diastolic volume was 136 ml/m², the volume at the time of aortic valve opening was 116 ml/m², the total stroke volume was 91 ml, and the regurgitant volume was 47 ml. In contrast to the localized basal reduction in V_{CF} observed in the patient with mitral valve prolapse, this patient demonstrated uniform velocity of fibre shortening in all chords; the mean V_{CF} fell within the normal range (Table), while peak V_{CF} appeared slightly below normal (Fig. 4).

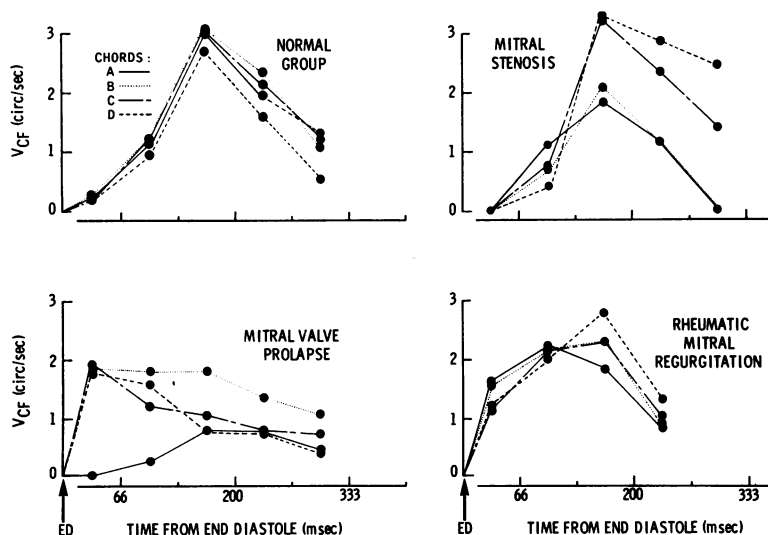


FIG. 4 The 'instantaneous' course of circumferential fibre shortening velocity for the normal group and for 3 patients with mitral valve disease are shown. Both mitral stenosis and mitral valve prolapse demonstrate a reduction in V_{CF} at the base. In both the prolapse and the rheumatic mitral regurgitation, significant fibre shortening occurred before aortic valve opening.

Fig. 6 shows the effect of alterations in loading conditions on mean V_{CF} , as well as on the mean velocities of the anterior and inferior radii, in 2 patients with coronary artery disease. Case 14 (inferior dyskinesia-akinesis) showed a pronounced reduction in the velocities of the anterior hemichords and a further increase in dyskinesia during an angiotensin infusion (increased afterload); ejection fraction fell from 52 to 35 per cent. In contrast, Case 15 (inferior akinesis with antero-apical hypokinesis) showed improvement in the velocities of the anteroapical hemichords and an increase in the ejection fraction (39% to 47%) after administration of nitroglycerin; however, the inferior wall motion remained poor.

Discussion

Analysis of circumferential fibre shortening rate in several chords of the normal left ventricle has revealed similar mean and 'instantaneous' velocities in all chords. Within a given normal ventricle any intracavitary pressure will be associated with uniform levels of wall stress throughout the ventricle, so long as the ratio of radius to wall thickness remains constant (Sandler and Dodge, 1963). If all areas of the left ventricle are subjected to similar inotropic influences and to similar loading conditions, uniform shortening velocities are to be expected. The values for mean and max V_{CF} found in this study are in general agreement with those pre-

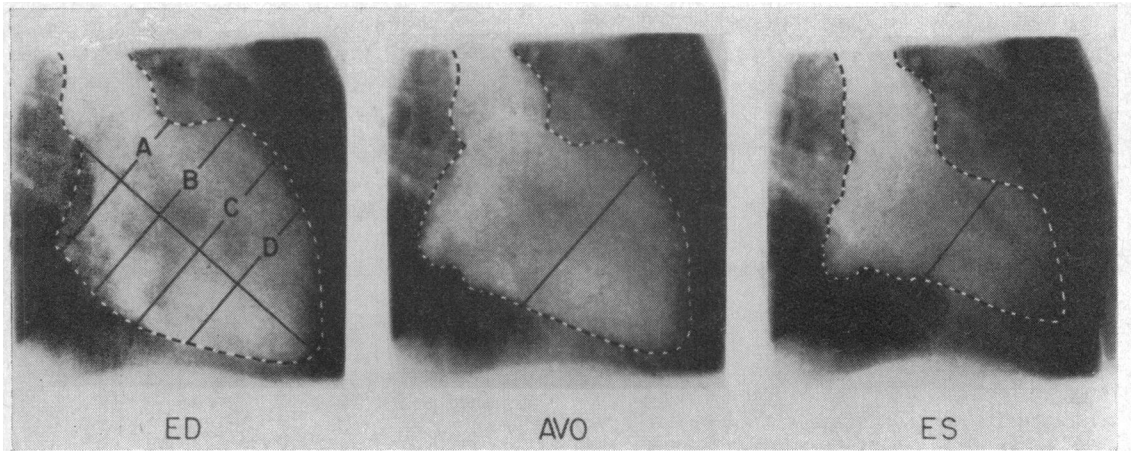


FIG. 5 Left ventricular cineangiogram from a patient with mitral valve prolapse. Chords A to D are identified in the end-diastolic (ED) frame. Significant mitral valve prolapse and fibre shortening occurred before aortic valve opening (AVO). ES = end-systole.

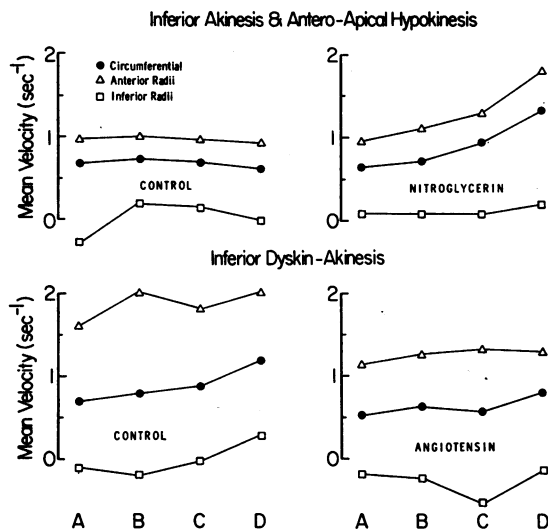


FIG. 6 Effects of nitroglycerin (top) and angiotensin infusion (bottom) on mean V_{CF} and normalized hemichord (radii) velocity in 2 patients with asynergy. Nitroglycerin resulted in improvement of the antero-apical hypokinesis with little change in the inferior akinesis. In contrast, angiotensin produced a reduction in the velocity of the anterior wall; paradoxical motion developed in inferior hemichords C and D. Dyskin = dyskinesis.

viously reported by Karliner and associates (1971b). Similarly, we observed a small amount of fibre shortening before aortic valve opening associated with a slight (3%) reduction in left ventricular

volume; these dimensional changes have been explained on the basis of redistribution of left ventricular volume towards the mitral valve area (Karliner, Bouchard, and Gault, 1971a).

Mean and 'instantaneous' V_{CF} were depressed in the areas of myocardium involved by abnormalities of contraction; however, when asynergy involved only one of two opposing walls, measurements of the velocity at the hemichords (or radii) provided a better definition of regional performance (Fig. 3 and 6). V_{CF} was normal at the basal chords (A and B) in the patients with apical asynergy. Of interest is one patient with apical hypokinesis who showed increased mean as well as max V_{CF} at chord B. This augmented contraction of the non-involved area has also been observed by others (Klein, Herman, and Gorlin, 1967; Hood, 1970). Though the reason for this localized increase in the velocity of shortening is not clear, several explanations may be postulated: hypertrophy of the non-involved myocardium (Bergmann, 1968), a rise in circulating catecholamines (Richardson, Woods, and Bagwell, 1960), or local metabolic readjustments (Gudbjarnason and Puri, 1969). In addition, it has been suggested that two determinants of end-diastolic myocardial fibre stretch are end-diastolic stress and end-diastolic compliance (Gaasch *et al.*, 1972). In this patient end-diastolic stress was increased (LVEDP = 18 mmHg). If compliance was normal in the non-ischaemic myocardium, end-diastolic muscle fibre stretch in this area may have been increased leading to augmented muscle function secondary to the Starling mechanism.

In one patient with asynergy, angiotensin (in-

creased afterload) produced a generalized reduction in the velocity and the extent of shortening of the non-akinetic myocardium, while in a second similar patient, nitroglycerin was associated with the opposite effect. Covell and associates (1973) have made similar observations in dogs suggesting that V_{CF} is sensitive to acute changes in afterload. Though a reduction in both afterload and preload occurs with nitroglycerin (Dumesnil *et al.*, 1973; Burggraf and Parker, 1974), other mechanisms, such as improvement in myocardial oxygen demand-supply relation, may be responsible for the observed changes. Pine and associates (1973) have observed improvement in wall motion after aortocoronary bypass surgery only in those asynergic areas that showed improvement with nitroglycerin during the preoperative evaluation. These findings suggest that some asynergic areas contain viable myocardium which is capable of improving its performance when loading conditions or oxygen demand-supply relations are altered.

It has been suggested that impaired left ventricular performance in rheumatic mitral stenosis results from a localized abnormality involving the posterobasal area of the left ventricle (Heller and Carleton, 1970). A reduction of mean and max V_{CF} confined to the base was seen in a patient with mitral stenosis who had a normal ejection fraction and a normal V_{CF} at the other chords. However, more generalized abnormalities of contraction have also been reported in patients with mitral stenosis (Holzer *et al.*, 1973). The aetiology of these abnormalities is not clear though residual effects of rheumatic myocarditis (Holzer *et al.*, 1973) as well as immobilization and atrophy of the posterobasal wall secondary to thickening of the valve leaflets and chordae tendineae (Grant, 1953; Heller and Carleton, 1970) have been suggested.

Angiocardiographic descriptions of the prolapsing mitral valve syndrome have focused primarily on the abnormalities of the mitral valve apparatus. However, additional information has indicated that the disorder is not limited to the mitral valve. Recent reports have described abnormalities of left ventricular contraction; Liedtke *et al.* (1973) found reduced mean V_{CF} at a basal chord in 9 patients with systolic click syndrome, two of whom had prolapse of the mitral valve shown angiographically. Scampardonis and associates (1973) have described 5 different patterns of left ventricular contraction, including a hyperdynamic, cavity obliteration type, but no segmental analysis of contraction was reported by them. In addition to a reduced mean V_{CF} at the basal chord, our patient with mitral valve prolapse demonstrated a significant reduction (22%) in the left ventricular volume

before aortic valve opening accompanied by an early peak in V_{CF} (Fig. 4 and 5). In this patient the early prolapse led to a partial translocation of ventricular volume into the posteriorly displaced leaflets. These changes in volume and V_{CF} before aortic valve opening were more dramatic than those observed in the patient with chronic mitral insufficiency. The absence of a true isovolumic period in this type of patient may limit the use of isovolumic indices of contractility. The patient with chronic mitral insufficiency demonstrated uniform velocity of fibre shortening in all chords; though mean V_{CF} and systolic ejection fraction were normal, max V_{CF} was slightly reduced (Fig. 4). Eckberg and associates (1973) have recently reported a group of patients with chronic severe mitral insufficiency, normal systolic ejection, and reduced V_{CF} . They have postulated that the rapid unloading occurring in these patients, caused by regurgitation before aortic valve opening, may help to maintain a normal ejection fraction in the presence of a reduced inotropic state. Though a good correlation between mean V_{CF} and max V_{CF} has been reported, occasional instances of discrepancy between the two measurements have been observed (Karliner *et al.*, 1971b).

The determination of V_{CF} at multiple chords, complemented when necessary by measurements of the velocity at the various hemichords, appears to be of value as a quantitative approach to the evaluation of disorders of regional contraction. Comparison among patients is possible since the velocity measurements are normalized. Potential uses of this technique include follow-up of patients with coronary artery disease, particularly those subjected to some therapeutic intervention. The sensitivity of V_{CF} to acute changes in afterload, however, may limit in some instances the usefulness of this measurement in quantitating segmental contractility.

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